The Measurement of Arterial Pressure in Man. I.—*The Auditory Method.*

By Martin Flack, Leonard Hill, F.R.S., and James McQueen.

(Received December 3, 1914).

From the Physiological Laboratory, London Hospital Medical College (London Hospital Research Fund), and the Pathological Laboratory, Aberdeen University.)

In a previous communication* we showed that when an artery, exposed in a living animal, is compressed in a glass compression tube full of water (Ringer's solution), the pulse, distal to the compression, is not obliterated until the pressure of the water is raised just above the systolic pressure of the blood in the artery, whereas when the same artery, placed on bone, wood or glass, is compressed by the bag of Leonard Hill's pocket sphygmometer, or by the armlet of the sphygmometer, so arranged that it does not embrace the surrounding pulsating tissues, the pulse is abolished by pressures under, and even much under, the diastolic pressure of the blood stream.

These facts are correlated with the manner in which the artery is compressed in each case. Enclosed in the compression tube the artery is compressed by the water equally in a circular fashion so that the rise of external pressure, up to the diastolic pressure, has no effect in producing deformation of the artery. Ultimately, when the compression becomes greater than the diastolic pressure, the artery flattens and changes to the oval shape during diastole. It is flattened during systole when the external pressure rises above the systolic pressure. When the carotid artery of the living animal is freed from the surrounding tissues and placed on a watch-glass and compressed by the bag of the pocket sphygmometer, or by the armlet so arranged as not to embrace the pulsing tissues of the neck, the oval deformation sets in at far lower pressures and is complete in relatively thin-walled labile arteries at pressures much under diastolic pressure. Consequently the blood flow is cut down by an external pressure less than diastolic to a mere ineffective trickle of blood, and the pulse is completely damped out. If a branch of the carotid artery distal to the bag were connected with a C-spring manometer, the record would show a progressive lowering of both the systolic and the diastolic pressure almost to zero, till with an external pressure much less than the diastolic pressure in the aorta the pulse was damped out and the blood flow became a trickle.

* 'Roy. Soc. Proc.,' B, vol. 87, p. 344 (1914).

Now MacWilliam and Melvin* have studied the behaviour of an excised artery compressed in a tube containing water with external pressures from zero up to diastolic pressure. They find that with pressures, say, from zero up to 50 mm. Hg, the distal manometers, systolic and diastolic, give practically unchanged readings. It is only when the external pressures become equal to the diastolic, or above it, that any great alteration in the distal manometer reading occurs. We find that this is so in the case of the carotid of a living animal enclosed in a glass compression tube and compressed by water. On the other hand, in the case of the same artery placed on a watch-glass, a much lower external pressure applied with the bag of the pocket sphygmometer is sufficient to obliterate the pulse.

The question now arises, how far do the conditions of an artery embedded in the tissues and surrounded with the tissue vessels resemble those of an excised artery enclosed in a compression chamber full of water? Were the facts demonstrated by this simple schema applicable, pressures from zero to 50 mm. Hg should have no effect upon the diastolic and systolic pressures in the brachial or radial artery distal to the point of compression.

But such a conclusion is contrary to every-day clinical experience. When an armlet is placed on the upper arm and the radial artery is felt at the wrist, and the pressure in the armlet is raised from zero to 50 mm. Hg, at some stage, varying under different conditions to be detailed later, the pulse beat at the wrist increases perceptibly in force. Accordingly we have here a seeming paradox, augmentation of the pulse produced by an external pressure rising from 0 to 50 mm. Hg—a pressure sufficient to deform an exposed artery lying upon bone and damp out the pulse, but which has no effect on an artery in the simple schema of MacWilliam and Melvin.

The phenomenon in question is obtained with a varying degree of facility in different people. In a patient whose systolic pressure was 130 and diastolic 80 mm. Hg, the increase in the force of the pulse beat became perceptible at the wrist when the external pressure in the armlet on the upper arm was raised to 35–40 mm. Hg. After taking exercise for three or four minutes, when the heart had been made to beat violently and the pulse-pressure† range increased in extent, a condition approaching to the findings in aortic disease, an increase in the force of the pulse beat became apparent with 10 mm. of external pressure in the armlet.

In a case of aortic disease with a large pulse-pressure range, the increase in the force of the pulse beat became apparent with 5–10 mm. of external pressure in the armlet.

* 'Heart,' vol. 5, p. 153 (1914).

† The difference between the diastolic and systolic pressures.

From a case of aortic disease having an aberrant radial artery we have taken tracings, using the Dudgeon sphygmograph with weight extension. The absence of the plethysmograph effect and of disturbance from venæ comites—there are none accompanying the aberrant artery—was thus secured. In the tracing (fig. 1) the external pressure in the armlet at the upper arm was, to begin with, 0 mm., then raised to 30 mm. The size of the initial pulse beat was regulated by the weight in the pan of the extension apparatus; some little skill is needed to secure the proper weight to show the phenomenon in a marked fashion.

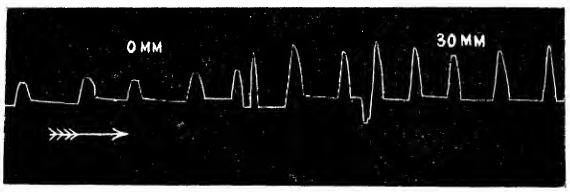

Fig. 1.

The tracing demonstrates that there is an increase in the systolic pressure in the radial artery as a result of compressing the upper arm; there is consequently an increase in the pulse-pressure range.

Corroboration of these tracings has been obtained by placing one armlet on the calf and another on the thigh of a boy. In normal boys we find the thigh reading to be higher than that of the calf.*

Boy.	Systolic pressure (horizontal posture).	
	Thigh.	Calf.
	mm. of Hg.	mm. of Hg.
I.	165	135
		140 when 60 mm. Hg was maintained in the thigh armlet.
		130 „ 0 „ „ „
II.	155	120
		135 „ 40—50 mm. Hg „ „
III.	105	80
		90 „ „ „ „

* In the case of two patients we have observed a systolic pressure considerably higher in the calf than in the thigh; *e.g.*, 175 thigh, 300 calf, upper part, and 225 mm. Hg lower part of leg. These differences of pressure obtained in both legs. It has been suggested to us that the higher reading in the leg may be due to protection of the artery from compression by fascinæ. It is difficult to see how this can come about, and we are at present unable to offer an explanation other than one based on the observations detailed in the following paper. The difference was certainly not due to resistance to compression on the part of the arterial wall, for the dorsalis pedis artery in these patients offered no unusual

The measurements of systolic pressure thus show an increase in the calf reading brought about by raising the pressure in the thigh armlet to 40–50 mm. Hg.

We find that the increase in the force of the pulse beat in the artery distal to an armlet coincident with the rise of armlet pressure from 0 to 50 mm. is correlated with the appearance of certain sounds audible with a stethoscope (Bowles' tambour form was used) placed at the elbow.

An armlet is placed on the upper arm and the stethoscope laid without pressure at the bend of the elbow; on raising the pressure in the armlet above systolic pressure and then lowering it gradually, clear sounds became audible, gradually getting louder; these are followed first by murmurs and then by loud clear sounds. At a certain level, which is taken as the index of the diastolic pressure, the loud clear sounds suffer a sudden diminution in volume and tone, to be succeeded by dull sounds; these may be audible much below diastolic pressure, *e.g.* 28–35 mm. or so.

These dull sounds, we believe, are due to the sudden tension of the artery and its branches produced by the impact of the systolic wave. This sets the mass of tissue enclosed in the armlet into vibration, the vibrations becoming audible to the ear as sounds. In the case noted previously these dull sounds appeared when the pressure in the armlet enclosing the upper arm reached 35–40 mm. Hg. At precisely the same level the force of the pulse beat could be felt increased while palpating with the fingers the radial artery at the wrist. When the subject took violent exercise the dull sounds became audible when the pressure in the armlet reached 10 mm. Hg, and increased in loudness with successive increments of this pressure.

In aortic disease, where the pulse-pressure range is high and the artery with each systole suffers considerable distension, the sounds are audible at the bend of the elbow under ordinary conditions. These sounds are at once increased in volume when the pressure in the armlet is raised 5–10 mm.; at precisely the same levels of external pressure the finger feels the pulse grow stronger, and the sphygmographic tracing confirms the sensory impressions from the fingers.

Therefore we conclude that a sound is given forth dependent on the pulse-pressure range of the blood stream which enters the armlet. If that range is high, *e.g.* in aortic disease, the systolic wave is big enough to cause the sounds to be produced under ordinary conditions. If the pressure within the armlet is raised 5–10–20–30–40 the systolic wave is reinforced by the

resistance to compression by the bag of the pocket sphygmometer. Further, it is difficult to suppose that the arteries in either leg should offer the same local resistance to compression and a resistance wholly different to that of arteries in other parts of the body.

increased tension of the arteries under the armlet and the sound becomes louder.

The bigger the pulse wave passing to the armlet, the ampler will swing the vessels under the armlet and the less the compression required to increase the force of the pulse wave in the radial artery at the wrist, and produce the dull sounds. We believe these sounds are produced by the impact of the systolic wave vibrating the tense artery and its branches, big and small. Consequently they should be independent of the blood flow. An artery ligated beats up to the point where the ligature is applied. Normally we feel pulses by closing the artery with the ball of the finger or thumb and feeling the impact of the systolic wave on the tip of the finger or thumb. To deform the artery with the finger a lower pressure than the diastolic pressure suffices. The finger deforms it just as the bag of the sphygmometer deforms an artery placed on bone.

It is possible to arrange an armlet on the upper arm and a second armlet immediately below the tambour at the bend of the elbow. Suppose the pressure is raised in the armlet to 30–40 mm. Hg, sufficient to call forth the dull sounds, and that then the armlet pressure below the bend of the elbow is raised far above the ascertained systolic pressure of the blood. This stops all effective flow in the artery at the bend of the elbow; the subsidiary branches of the main arterial trunk rapidly fill up, as all exit for the blood is blocked in the distal area by the compressing armlet placed below. Yet the dull sounds persist. Further, these sounds become more audible as the blood flow is stopped. That is exactly what one would expect if the sounds are due to sudden tension. The artery and all its branches become tenser above the block. The whole kinetic energy of the pulse spends itself in striking the tense labile artery and its branches. The whole mass of tissue under the armlet—permeated with blood vessels—is struck by the pulse.

We find that the phenomenon of an increase in the pulse force in the radial artery at the wrist is often felt best at the first examination of the patient with the sphygmometer. The excitement produced by examination increases the force of the heart, and the high crest of the pulse wave reaching the tissues compressed by the armlet becomes reinforced: an increase in the force of the pulse beat is thus felt easily with a low pressure within the armlet. As the excitement subsides, and the pulse beat becomes normal in its range, the level of pressure at which the increase is felt becomes higher.

As regards the production of the loud sounds and murmurs heard on passing from diastolic level to systolic level, it is possible to separate the element of sound due to pure tension of the arterial wall and the element of sound which requires a flow of blood.

Suppose one raises the pressure in the armlet on the upper arm to 100 mm. and so develops at the elbow the loud characteristic murmur. If one then raise the pressure in another armlet placed below the auscultating tambour, to well above the known systolic pressure in the artery, this murmur disappears but a sound synchronous with each pulse beat appears in its place. This is the dull sound due to the sudden tension of the arterial wall, a sound independent altogether of those vibrations which are set up in the arterial wall by that inrush and outrush of the blood which is synchronous with the crest of the systolic wave. On lowering the armlet pressure to the level at which the outflow of blood regains sufficient velocity the characteristic murmur returns. We conclude, therefore, that stoppage of the blood flow by the lower armlet while the pressure in the upper armlet ranges up to systolic pressure, cannot prevent the occurrence of sounds, though it leaves their quality changed. The sounds due to vibrations set up by the sudden in and outrush of blood disappear; the sounds due to the periodic sudden tension of the arterial walls persist.

As we have said before, in an artery placed on bone or glass and compressed with an armlet (the armlet not embracing pulsing tissues), or with the bag of the pocket sphygmometer, the pulse is obliterated by pressures below diastolic pressure.

By the reinforcement of the pulse in the vessels of the tissues which are enclosed by the armlet or bag, used in the ordinary way, these critical pressures are successfully passed, and the normal process of arterial deformation proceeds at the proper level. So accurate systolic blood-pressure measurements, both auditory, tactile, and visual, become possible. On this mechanism—the conserving effect of the tissue vessels on the pulse—depends the accuracy of the auditory method of estimating the diastolic pressure. Without this mechanism the diastolic level would be too low. Thus we have found it so when the bag of the pocket sphygmometer, or armlet, used so as not to embrace pulsing tissues (as described in a previous communication, *loc. cit.*) is applied to the aberrant radial. But when the armlet is applied round the arm then the diastolic auditory index, as heard in the aberrant radial, comes much closer to the truth.

It has been noted by MacWilliam and Melvin, and others, that sounds can be produced which are audible at the brachial artery at the elbow when finger pressure is applied to the brachial artery in the arm. Hill, McQueen, and Flack (*loc. cit.*) have shown that finger pressure applied discretely to any artery, brachial or radial, deforms the artery in a precisely similar manner as does the bag, or armlet (used with the box so as not to embrace pulsing tissues), when applied to the aberrant radial. The pulse is damped out below

diastolic pressure. Consequently the sounds produced by finger pressure on the brachial artery, while they somewhat resemble in quality the normal sounds produced by armlet pressure, do not show a perfect similarity to these. Thus, suppose we obliterate the brachial artery with the finger, on releasing it gently we hear for a very short period clear sounds followed by murmurs. These murmurs are not in our experience followed by clear sounds and then by dull sounds, as is the case when the armlet embraces the upper arm. It is obvious that on slightly releasing the occluding pressure the blood flows in jets into the artery, which is relaxed below the seat of compression. Hence the first clear tension sounds. When the artery becomes oval in shape, the clear sounds are dulled by the murmurs. When the artery becomes circular the pulse wave does not suffice to make tense the now patent artery and produce the dull sounds. The finger does not obstruct the peripheral outflow and bring into play the reinforcement due to the vessels of the tissues.

If we place an armlet distal to the position of the stethoscope, and by the pressure in this armlet obstruct the blood flow, then on compressing the brachial artery with the finger, only clear sounds are heard for a short period as the artery is compressed and released. The murmurs vanish, clear sounds take their place, and the range of sounds is short. As we have pointed out, the whole range of sound is dependent on the resonating effect of the vessels in the tissues surrounding the artery. This resonating effect is absent when an artery is discretely occluded by the finger so that surrounding tissues are not included.

If an armlet is placed on the upper arm and the external pressure raised till the clear sounds are produced, just below the systolic level, then if the artery be occluded by the finger between the armlet above and the tambour of the stethoscope below, all sounds vanish. Supposing we place the tambour under the lower part of the armlet, then compression of the artery immediately distal to the edge of the armlet does not abolish the sounds. They become clearer, because the energy of the pulse spend itself on the mass under the armlet; the obstruction further tightens up the vibrating drum.

The sounds are abolished when the finger occludes the artery between the armlet and the tambour; first, because the artery is not distended by the systolic phase of the pulse wave; secondly, because the sounds produced under the armlet are not now conducted by the fluid column of blood in the artery.

Suppose we place the stethoscope under the lower part of the armlet, that is just above the occluding finger, then the sounds are audible just up to the systolic pressure of the blood, *e.g.*, 110 mm. Hg. But if the stethoscope is placed exactly under the upper edge of the armlet, sounds are audible up to,

say, 200 mm. Hg. in the armlet, a pressure far above the systolic pressure of the blood. It is clear that when an armlet is placed on the upper arm and the pressure raised above systolic pressure, the systolic wave must meet a sudden check at the upper part of the armlet, hence the arteries are here made tense and the sounds produced by their sudden tension are heard. It is possible under these conditions (MacWilliam and Melvin* have observed it in one case) that the sound, if exceptionally loud, may be conducted by bone below the armlet. Their case was one of aortic regurgitation in a healthy student—a good athlete.

Conclusions.

1. In the measurement of arterial pressure by means of the armlet and sphygmometer the auditory method gives clear indices of systolic and diastolic pressure.

The auditory indices are (1) loud throbs heard in the artery below the armlet when the compression is lowered just below the systolic pressure; (2) a sudden diminution in the sounds when the pressure falls just below the diastolic pressure. We find that these indices depend on the pulsatile flow of blood in and out of the part compressed by the armlet; the artery is deformed by the compressive force and its wall swings out and in when the pulse wave strikes the deformed part. When the flow of blood into the limb peripheral to the armlet is obstructed the throb is no longer heard, but is replaced by dull sounds caused by the pulse striking the tense arterial wall.

2. Dull sounds are heard, under ordinary conditions, when the compression is reduced below the diastolic pressure. Such slight compression gives occasion to the pulse to produce the dull sound, by obstructing the venous outflow, and thus raising the diastolic pressure in the arteries and the tension of their walls.

3. The bigger the systolic wave the less compression is required to make audible the dull sound.

4. The accuracy of the auditory method depends on the conserving effect which the tissue vessels have on the arterial pulse when the arm is compressed.

5. The method cannot, therefore, be used to give accurate measurements in the case of an artery lying on bone and unsupported by tissue vessels such as the aberrant radial or dorsalis pedis.

6. Accurate readings can be obtained from these arteries where they lie embedded in tissues, and the reinforcing effect of the tissue vessels comes into play.

* 'Brit. Med. Journ.,' 1914, A, p. 697.

7. Clinicians know that the pulse in the radial artery becomes more forcible when they begin to compress the arm. At the beginning of compression of the arm, the armlet, by obstructing the venous outflow and making tenser the arteries in diastole, improves the conduction of the systolic wave. The pulse in the radial artery, therefore, becomes reinforced. The dull sound and the reinforcement of the pulse are due to the same cause.

8. Evidence has been obtained then, by experiments on man, of the effect of increased tension of the arterial wall (lessened lability) on the conduction of the crest of the systolic wave.

The peripheral conditions affect the lability and the pressure readings.

The Measurement of Arterial Pressure in Man. II.—*A Schematic Investigation.*

By Martin Flack, Leonard Hill, F.R.S., and James McQueen.

(Received December 3, 1914.)

(From the Physiological Laboratory, London Hospital Medical College (London Hospital Research Fund), and the Pathological Laboratory, Aberdeen University.)

MacWilliam and Melvin* have demonstrated in the case of the excised artery—compressed in their schema—that a compressing force which was not sufficient to obliterate the pulse caused a great fall in the manometer, which they placed distally to the compression tube. To cite an example, the entering pressures in the proximal manometers were: systolic 178 mm. Hg, diastolic 118 mm. Hg. A compressing force of 140 mm. Hg caused a great fall in the distal manometers—systolic became 42 mm. Hg, diastolic 22 mm. Hg. We find that the artery, under these conditions, is flattened during diastole, and the inflow during systole is not of sufficient duration to maintain the distal pressure, supposing the resistance to outflow is unchanged. If the resistance to outflow is increased, no such distal fall of pressure occurs.

Their schema differs in essential points from the conditions which pertain to an artery embedded in living tissues and encircled by an armlet. The pressure within the armlet at first does not deform the artery, but expresses blood from, and increases the peripheral resistance in, the mass of tissue it

* 'Heart,' vol. 5, p. 153 (1914).